Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION
50 WEST 50TH STREET, NEW YORK, N. Y.

DR. SAMUEL A. LEVINE, Boston, *Editor*
DR. MARSHALL N. FULTON, Boston, *Associate Editor*

Vol. III December, 1934 No. 12

ACUTE BACTERIAL ENDOCARDITIS

In discussing this particular type of endocarditis it might be well to introduce the subject with a word of explanation as to the terminology that has been used in the past and that which is being used today. Up until comparatively recent times the terms malignant or ulcerative endocarditis have been employed to describe acute bacterial endocarditis. The first adjective, malignant, is appropriate in the sense that the disease runs a rapid and fulminating course quickly terminating in death. This term has the misfortune, however, of being so intimately connected with new growths that discarding it is justifiable. Ulcerative applies to the pathology that is produced by the organisms and it is not inappropriate. On the other hand, the present day tendency in diagnosis is to stress etiologic factors rather than to accentuate the morbid changes that occur in disease. The adjective bacterial, which is used in modern glossology to qualify the word endocarditis, is frequently followed in parenthesis by the further qualifying adjective, infective, which in turn has replaced the term infectious used some years ago. The most accurate terminology that can be employed would be to speak of the disease as acute bacterial, in parenthesis naming the causal micro-organism and then using the main term endocarditis. Thus more accurate information would be obtained from the study of bacterial endocarditis if it is expressed acute bacterial (pneumococcic) endocarditis, or (streptococcic) endocarditis, than merely to express the disease as acute bacterial endocarditis without further indication as to the etiologic agent.

Acute bacterial endocarditis does not include the acute expressions of rheumatic endocarditis. This is mentioned to obviate the confusion that sometimes arises when there is a discussion of this disease. Acute bacterial endocarditis arbitrarily is separated from the subacute type by a time period usually considered to be not more than two months; the duration of the disease, from the onset to death, very rarely exceeds a few weeks. An acute endocarditis persisting for a longer time than this period usually is labeled subacute. While this is an artificial division yet it fits in well with clinical experience in that very distinct differences exist between the type of endocarditis, subacute bacterial, produced by Streptococcus viridans, not only in its duration but also in its expressions, from that which is produced by other organisms of the cocci group which are responsible for the fulminating type of endocarditis.

ETIOLOGY

The figures compiled by the late Dr. William S. Thayer are very generally accepted as indicative of the frequency and the incidence of acute bacterial endocarditis. They are (a) hemolytic streptococcus, 55 per cent; (b) pneumococcus, 13 per cent; (c) Staphylococcus aureus, 12 per cent; (d) gonococcus, 11 per cent and (e) miscellaneous organisms making up the remainder. These figures are in accord with other observers of a considerable number of cases. It should be noted that the coccal group of organisms overwhelmingly predominate as the agents responsible for the endocarditis. The organisms enter the blood stream, subsequently to attack the valve from a variety of primary loci. A pneumococcic lung infection, a puerperal sepsis, an infected wound, an osteomyelitis, even furuncles may be the primary responsible locus from which the disease starts. Manifestations of these several types of bacterial endocarditis vary not inconsiderably, but the picture taken as a whole does not differ to any very great extent.

SYMPTOMS

Acute bacterial endocarditis in the majority of instances is overshadowed, at the onset at least, by the disease which is producing the infection. Pneumonia, puerperal sepsis, gonorrheal rheumatism, meningitis, and septic infections may produce symptoms which befog the diagnosis of involvement of the heart. This applies particularly to those cases in which there is an obvious septicemia. The high fever, most generally septic in type but often maintained, or extreme prostration, delirium or coma all hide, obscure and becloud the underlying valvular involvement. The disease is essentially, moreover, a septicemia and it is rather an academic question as to whether or not heart involvement has occurred. Ultimately the failure to recover from a pneumonia, for example, the development of evidence of focal suppurative processes and a great accentuation or increase in intensity of a previously soft systolic mitral or aortic murmur will establish the diagnosis.

Fever is invariably present at the onset; usually it is continuous with rather marked remissions and intermissions. The remissions are brief and transitory. With the elevated fever there is usually a chill on and off during the course of the septicemia. The pulse rate is rapid, usually more so than the severity of the fever would account for. Embolic phe-

nomena are extremely common, often terminating as metastatic abscesses. These abscesses are particularly common in Staphylococcus aureus infection, which, after all, is a true pyemia. Enlargement of the spleen is a common finding but not as frequent as occurs in the subacute type of endocarditis. The spleen is frequently not noted to be enlarged until late in the course of the disease. Arthritis is another common complication or expression of the disease, particularly likely to be present when there is a gonorrheal septicemia. The clinical expressions represent, with the exception of petechiae and the embolic phenomena, nothing which would point directly to involvement of the valves. The laboratory examinations likewise indicate nothing much more than a septic process with blood stream infection. The leukocyte count is almost invariably high, with the usual granulocytic formula of sepsis and the blood culture is sooner or later positive. The urinary picture is that of a febrile urine with albuminuria and casts, and in a few instances red blood cells may be found to indicate a focal, or hemorrhagic nephritis. An anemia will develop but probably because of the shorter duration of the disease cannot be compared in severity to the anemia that develops in subacute bacterial endocarditis.

Physical examination of the heart, as well as the autopsy findings, indicate that about one-half or less of the patients have had previously existing valvular or congenital heart disease. There occurs in a certain number of instances a change in the character of the valvular murmur which may be the ultimate criterion for the diagnosis of endocarditis. Unfortunately the ulcerative process on the valve does not by any means produce a murmur which in itself is indicative of the extensive destruction of valve tissue that may be disclosed at autopsy. A word might be said as to the valves most commonly affected. Usually it is the left side of the heart which is attacked and the aortic or the mitral valve seem to be about equally susceptible to infection by the pathogenic organisms producing the disease. The right side of the heart is by no means inviolate. In gonorrheal endocarditis, for example, in Thayer's series the left side of the heart was involved in 12 instances, the right side 5 and both sides 4. While pericarditis is present in about one-fifth of the cases, the ante mortem diagnosis is extremely uncommon. The same statement may be made in regard to the acute myocarditis which likewise is an anatomic rather than a clinical observation. Physical evidence or evidences of either a primary focus or secondary foci are such as would be expected irrespective of the cause producing the condition. Thus the terminal meningitis of pneumococcal endocarditis is recognized by the usual signs of meningitis. An empyema likewise is appreciated by the common signs of pus in the pleural cavity. Pneumonia or venous thrombosis is similarly recognized by its local expressions and the same thing applies to arthritis. Cerebral symptoms may be part of the general septic features or they may be the result of endocardial thrombi which have lodged in the brain and which may produce a single abscess or multiple brain abscesses.

An electrocardiogram is of no substantial assistance in the interpretation of the heart involvement, though arhythmias are relatively frequent.

A comparison between bacterial endocarditis, acute and subacute, hardly seems necessary except in a very broad way. In the first place the lesions of acute endocarditis are truly ulcerative and destructive without evidence of, or attempt at repair, whereas in subacute endocarditis there is a tendency for reparative processes to occur, in fact, they frequently do. There is, in subacute bacterial endocarditis, a disposition for the wall of the endocardium, the aorta or pulmonic artery to be involved by extension of the valve vegetations which more acute forms do not show. Secondly, the duration of the disease and the severity are quite unlike in the two conditions. Thirdly, the prognosis is bad in both but recovery does take place in the subacute type, whereas it does not except as a medical curiosity in the acute type. In both of these two types the infecting organisms are implanted in the majority of instances on previously diseased or congenitally distorted valves but more frequently so in the subacute than in the acute type. Embolic phenomena and splenomegaly are more frequent in subacute endocarditis than in acute, but on the other hand abscesses, septic infarcts, are common in the staphylococcic, pneumococcic and streptococcic infections, rare in the gonococcic and extremely uncommon in viridans infections.

COMPLICATIONS

The phenomena produced by emboli to the brain, to the kidney and to viscera have been mentioned. These are by far the most common and most important complications that occur in this condition. The anemia is relatively slight but every once in a while there develops what is apparently a severe blood dyscrasia with generalized hemorrhages in the skin and mucous membranes of the body.

CAUSE OF DEATH

The patients die in most instances as a result of the severe septicemia, although more than 50 per cent of the pneumonic cases die of meningitis. In the other types the septic complications may be the immediate cause of death. Heart failure ensues in a small proportion of cases. Death may be very sudden, as when an embolus to the brain or to the coronary vessel occasions it. Usually there is a relatively gradual exhaustion from the overwhelming toxemia.

TREATMENT

There is no adequate treatment for the condition of acute bacterial endocarditis. It would seem logical to employ anti-pneumococcic or streptococcic serum in the pneumococcic and streptococcic cases, but, practically, the results have been entirely valueless. The use of vaccines is not indicated and the intravenous administration of dyes such as mercurochrome or gentian violet in the staphylococcic infections likewise seems to be useless. The very exceptional and occasional patient who recovers apparently does so by his own inherent ability to resist and conquer the disease. Transfusions may be of some assistance in tiding the patient over for a period of time. They should be small, not over 250 cc. of blood, and repeated frequently, rather than large transfusions, which may overtax the injured heart. General supportive measures should be carried out and the patient should be given a diet adequate in calories, vitamins and minerals. If delirium or coma prevents the patient from eating he should be fed through the nasal catheter, giving food of high caloric value and at least 1500 cc. of fluid per diem.

J. H. MUSSER, M.D.
New Orleans, La.